TREATMENT OF INHALATION INJURY

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The expression "inhalation injury," in English terminology, includes conditions as different as those due to soot inhalation, barotrauma lesions or respiratory burns. Our point of view is that "inhalation injury" represents only one instance of a more general condition called "Primary Respiratory Lesions."

This expression "Primary Respiratory Lesions" includes all the direct pulmonary lesions induced by the causal agent itself at the time of the insult.

These direct lesions include:

— Macroscopically visible lesions seen by fiberoptic bronchoscopy:
- Burns of the respiratory tract brought about by thermal or chemical sources through irritating and caustic action in the tracheal or bronchial mucosa.
- Soot invasion, resulting at least in atelectasis and infections, even in burns when the soot is hot or irritating.

— And there are bronchiole and alveolar injuries not visible with endoscopy.
- Thermal and especially chemical inhalation injuries. Direct damage to the surfactant is probably implicated.
- Barotrauma injuries (pulmonary blast injuries) affecting the alveolocapillary membrane with probable indirect destruction of the surfactant.

The foregoing injuries represent the hidden part of the iceberg. But all of the lesions I have mentioned often occur in an already pathologic lung and create a vicious circle which perpetuates the respiratory trouble.

Indeed, in any severely burned patient, apart from any direct pulmonary assault, there exists a set of indirect alveolar lesions, subordinated to the skin burn itself, which is common in patients with extensive burns. This we call the "Burned patient's lung."

It consists of a particular form of shock lung, a real Da Nang lung, due to mass liberation of catecholamines and vaso active substances (serotonin, kinins) to which is added a liberation of intestinal toxins because of ischemia. Since these toxins are not detoxified by an ischemic liver, they reach the pulmonary micro-circulation in which hypoperfusion, acidosis and local hypoxia have occurred. This leads to failure of surfactant regeneration. Clinically, the burned patient's lung shows a lesional edema with hypoxia and a decrease in bacterial clearance, aggravated by burn therapy and its massive perfusions.

On the physiopathologic level, in addition to extrinsic constraint to the mechanics of breathing due to deep burns on the chest or on the neck, it is necessary to roughly distinguish four lesional levels, each with its own characteristics, but whose pathologies are often intertwined.

1st The laryngeal level: This includes simple upper tract obstruction, by local edema, with an air-way limitation.

2nd The bronchial level: The presence of a bronchial burn induces an

edema and a bronchoplegia with a decrease in the bronchial lumen, desquamation and secondary hemorrhages follow, resulting from the separation of eschars which can obstruct the airway; bronchial drainage is impossible to carry out because local defenses are broken down, especially mechanical ones, hence obstruction and infection occur.

3rd The bronchiolar level: Bronchiolar injuries cause an increase in the closing volume (CV) because of edema, the CV/Functional Residual Capacity (FRC) ratio increases and some areas are shunted.

4th The alveolar level, where edema and extravasation induce a decrease in the functional residual capacity. The CV/FRC ratio is above 1 and some airways remain closed during a part of the ventilatory cycle; there is therefore a shunt effect. These variations of the closing volume and of the FRC are usually linked; they worsen mutually and constitute a key problem. It is possible to correlate these two parameters CV and FRC with the alveoloarterial difference in the partial pressure of oxygen ($PAO_2 - PaO_2$).

On the biologic level, one sometimes sees hypercapnia, due to endogenous hyperproduction of CO_2, or to gradual exhaustion of the patient or to associated pathology. However, the most frequently observed disorder in patients with Primary Respiratory Lesions is actually a hypoxia with a normo or hypocapnia.

Very likely, a 5th lesional level exists which is the capillary itself (Venus et al). By primary or secondary damage, it affects the Ventilation/Perfusion Ratio.

Therefore, beyond the bronchial level all lesions contribute to the formation of the white radiologic lung with hypoxia. The therapy of the skin burn itself may not be unconnected and may aggravate the pulmonary injuries.

The 1st question which arises is therefore the following: *Does the presence of PRL make it necessary to modify the usual therapy of the skin burn?*

A. Two aspects of vascular filling must be considered: *Quantitative and Qualitative.*
   1. *Quantitative:* It is necessary to choose between two risks.
      a. If too much fluid fills the blood vessels, pulmonary injuries will be worsened.

Peitzman, Shires and Curreri have shown that, actually, patients with parenchymal inhalation injury already have elevated extravascular lung water volumes.

      b. If too little fluid is supplied, the state of shock will be perpetuated with a worsening of skin injuries and of renal and other organ insufficiencies. Moreover, in underfilling, some techniques of efficacious ventilation are impossible to use. Hence, it is necessary to apply an optimum pressure of perfusion. Either pulmonary capillary pressure or cardiac output or myocardial function can be used to monitor hemodynamic flow. If we use only the PCP values to monitor fluid volume, and if these are low, we are inclined to increase the pressure. However, this may not be desirable because the increase in cardiac output does not correspond to the increase in the ventricular pressure, but with the *increase in ventricular volume.* Hence, we do not know the Pressure/Volume ratio.

It is also difficult to determine the optimum perfusion volume by measuring only the cardiac output. Hence it seems more rational to measure both the pulmonary capillary pressure value and the cardiac output (or the left ventricular systolic index) in order to regulate vascular filling and to obtain the highest efficiency with the lowest pressure.

We find it is useful to carry out a vascular filling test and if the filling is correct but if the heart is not efficient, indicated by an infranormal curve, which means a latent myocardial incompetency, it is advisable to quickly resort to an inotropic drug, such as Dopamine chlorhydrate. Whatever the method used, the arterio-venous difference in O_2 (DAVO2) can be calculated to determine the efficiency of tissue oxygenation.

2. *Qualitative:* As for the qualitative aspect of fluid input, it is a question of the choice between colloids and crystalloids. The synthetic colloids can be excluded, for they do not provide proteins, or immunoglobulin, or coagulation factors. Holleman, et al, have presented data which strongly support the inclusion of colloid in the resuscitation of severely burned patients: with colloids, plasma oncotic pressure is higher, PaO_2 is increased and lung water is lowered. These data do not agree with Tranbaugh's conclusions. However, Shoemaker has shown that in terms of volume, less colloid than crystalloid is needed to obtain the same hemodynamic result. The issue is still unresolved.

B. *Drugs Used In Burn Therapy*

The presence of primary respiratory lesions does not have a large influence on the choice of drugs. Anti-platelet aggregation agents, which seem to us already necessary in extensive skin burns ▷ 50% are even more advisable in the PRL, for we know that intravascular coagulation is a factor in the appearance of injuries encountered in Adult Respiratory Distress Syndromes.

C. *Local Treatment*

Apart from the use of mafenide, which can cause metabolic acidosis leading to hyperventilation, the alternative treatment is the early excision-graft. For me it is the method of choice except in patients with PRL, since surgery even with good anesthesia induces a postoperative reaction which may worsen lung injuries. This is true in patients with hemodynamic instability who cannot tolerate wide hemodynamic swings; in such patients, any intervention seems to us to venture too far. But on the other hand, if pulmonary disorders disappear quickly, a semi-early excision-graft may be contemplated on the 5th or 6th day, when the problems of the so-called "acute-phase" are about to be solved. I say, in passing, that as far as the fluidized air bed is concerned, we personally proved that it does not decrease the healthy subject's respiratory performances and for the burned patient with PRL the issue is a red herring once they are artificially ventilated.

Having defined the treatment of the burn itself, the second question which arises is: *What is the specific treatment of PRL?* Common sense dictates the course of therapy. The solutions for some cases are simple, for example:

- Escharotomy for chest or neck skin injuries which constrain breathing.

- Simple insertion of a tube to protect the airway in patients with an obstruction caused by edema of the upper airway.

Other cases are not so simple, such as "when does a patient need ventilation?"

If the patient arrives in respiratory distress, the question does not arise. It is the same as for cases of carbon monoxide poisoning, which should be looked for routinely.

One must not wait for clinical and biological signs of respiratory distress since experience shows that, with time, conditions of ventilation are worse, especially dangerous hemodynamically will be the PEEP levels required by the patient. Ventilation then is strongly indicated and must be used if there are:

- Macroscopic lesions,
- If there is initial hypoxemia with PaO_2 under 70 mm Hg and if it is poorly corrected by use of a nasal tube for delivering oxygen,
- Or if indicated by Philips' criteria,
- Or if there is likelihood of alveolar injuries: here it is necessary to rely on anamnesis which can be very important. For example, if an accident occurred in a closed space, if there was combustion of synthetic materials near the patient, or if there was an explosion. In these last two cases, it is useful to underline the importance of searching for toxic products in the blood, especially cyanide and thiocyanate. Indirect signs of pulmonary blast, such as tympanic damage are also frequently observed when looked for. It is better to insert a tube and to ventilate too much than too little. When foamy secretions occur, even an acute lung edema, it is often too late to operate effectively.

Another difficult question is how the patient will be ventilated, that is, which route of entry and which technique will be chosen?

If the patient's face is burned, the mask cannot be used. Besides, it is inadequate in practice to insure good control of ventilation. There remains the choice between tube insertion and tracheostomy. Several workers have shown that for obvious reasons it is not advisable to carry out a tracheostomy in a burned area. Moreover, the decrease in resistance to expiration caused by tracheostomy reduces the natural PEEP effect which is beneficial. We insert tubes nasally using a low pressure cuff endotracheal tube, and we monitor the pressure of the cuff. In order to avoid a tracheal infection, we clean the respiratory tract before tube insertion with a gauze tent impregnated with naphtazoline and xylocaine which provides an anesthetic effect, vasoconstriction and disinfection. The tube is changed when needed. As fas as choice of ventilation technique is concerned, it is in fact easy, and I'll describe to you here what we do in my Burn Unit. The presence of typical respiratory distress directly calls for controlled ventilation (IPPV or CPPV). In other cases, patients with injuries diagnosed by endoscopy, or by clinical, biological and radiological signs indicating the presence of PRL, tube insertion is systematic and we base the choice of the technique of ventilation on the study of the static thoracic compliance curve because finally the only real choice amounts to the determination of the modalities of expiration. The problem is a PEEP or not a PEEP? The compliance

curves are obtained from sleeping and curarized patients. The pressure/volume ratio permits determination of two types of curves: the first is a straight curve or upwards convex curve, corresponding to a normal or increased FRC. For these patients a positive pressure at the end of expiration is not justified since there are no additional alveoli to enlist. Since pulmonary status can change, these curves must be done every day for three days. The second is a sigmoid curve, the slopes of which determine the compliance with:

- A lower part the slope of which is very much reduced (low compliance) because of the closure of the bronchi and alveoli,
- A medium part of increased slope which indicates an increase of compliance due to opening of collapsed alveoli,
- A higher part with a decreased slope indicating a very high pulmonary volume (low compliance) because of the overdistension of the well ventilated alveoli. In this case, it is permissible to try to increase the Functional Residual Capacity by applying a PEEP which allows the patient to ventilate not where compliance is low but where it is high, represented by the medium part of the curve, insuring a better distensibility and corresponding to the enlistment of more alveoli. A PEEP which is too high would lead to ventilation reflected by the higher part of the curve where the compliance is again decreased and where the risks of a pneumothorax are higher because of the alveolar overdistension. Therefore, it is necessary to choose the appropriate values.

A third difficult issue is which level of PEEP to choose? The choice of the PEEP level must take into account favorable effects of the PEEP which result in an improvement of the patient's ventilation, and an increase in the FRC above the critical level of bronchial closing. It must also take into account the pernicious effects linked with the increase in the average intrathoracic pressures, responsible for a constraint of the venous return, of a decrease in the transwall pressures of cardiac filling and of a decrease in the cardiac output.

Several methods have been proposed since Ashbaugh, 15 years ago, proposed the "Positive Expiratory Pressure":

- Suter, et al, have proposed the level needed to obtain the best transport of O_2 (Q_2 x CaO_2) for an optimal level of PEEP.
- Kirby, et al, have chosen a level needed to obtain a QS/QT ratio under 15% as the best level of PEEP; this method reaches to levels up to 50 cm H_2O, which are dangerous.

It is possible to proceed by trial and error to progressively increase the level of PEEP to obtain a PaO_2 above or equal to 70 mmHg with an FiO_2 under 50%. This method, which tries to lower the FiO_2 of the inspired gas to a level assessed as non-toxic for the pulmonary parenchyma, may lead to a higher level of PEEP than the optimum one.

As for us, we have opted for prophylactic therapy which consists of avoiding the evolution of pulmonary injuries by trying at the start to obtain the level of PEEP which enlists enough alveoli to clear all or a part of the shunt.

If we compare the values of the shunt QS/QT and those of the pressure/volume curve with an increased PEEP level, we notice a close relation

between the inflexion of the PV curve and degree of removal of the intrapulmonary shunt.

Within several patients, the break in the initial part of the PV curve corresponds to a median pressure in the airways, of 9 cm H_2O. When the PEEP is set at this value or just above, we notice an important decrease in the intrapulmonary shunt QS QT inducing a sudden improvement of the PaO_2 which seems to correspond to the simultaneous opening of a great number of alveoli previously collapsed.

Thus the best level of PEEP seems to us to be equal or just above the pressure observed at the first point of inflexion, corresponding to the limit of the closing volume.

The increase in the FRC which is obtained in this way, elevates the FRC above the closing volume, and the CV/FRC ratio is under 1.

An hyperoxia test induced by $FiO_2 = 1$ ($O_2 = 100\%$) for 20 minutes does not seem dangerous for us and allows us to see without a Swan Ganz catheter that the shunt has been decreased (the PAO_2/ PaO_2 gradient must be under 300).

In the absence of compliance measures, we directly set the PEEP level between 10 and 15 cm H_2O, being aware that below 10 we are inefficient and above 15 it is often dangerous.

The last point to discuss is the choice in the means of PEEP.

The level of PEEP which efficiently enlists alveoli being determined, the best ventilation consists of allowing the patient to breathe spontaneously. It is the CPAP which insures a low average thoracic pressure and which has therefore little hemodynamic repercussions. Moreover, there is a no inverse relationship between blood flow and alveolar oxygenation. It is therefore the best means of ventilation.

However, it can't be used in patients with an associated polyvisceral deficiency. CPAP itself can exhaust the patient and therefore requires a rigorous clinical and gasometric surveillance.

We stop CPAP under the following conditions:

- A polypnea above 40/mn,
- A closing volume under 300 ml,
- A hypercapnia above 50 Torr,

and we change to CPPV ventilation which has undesirable effects (maximum hemodynamic repercussions and inverse relationship between blood flow and alveolar oxygenation). In order to avoid pressure swings, it is then important to have auto-trigger breathing.

In addition to these two methods of ventilation, there is another: IMV/PEEP, whose hemodynamic tolerance is comparable to the CPAP, as long as the frequency of mechanical inflations remains low (2 to 6/min) and the IMV is well synchronized with breathing. When a level of PEEP is thought to be good, it is necessary to maintain it with a favorable hemodynamic volume expansion and use of inotropic drugs rather than decreasing the PEEP value because hemodynamic repercussions are too serious.

Once methods of ventilation are established, one can use three therapeutic adjuvants to treat PRL: — Drugs — Fiberoscopy — Kinesiotherapy.

The advisability of using drugs for pulmonary disorders is not always obvious. We do not advise the use of corticoids for many reasons: a simple

dose is inefficient and may be harmful. High repeated doses contribute to sepsis in patients with decreased defenses and finally steroids have no beneficial effect upon arterial blood gases or pulmonary dynamics. In the two groups treated by Moylan, the steroid treated group had a mortality rate that was four times that of the placebo group. Moreover, as the Fort Sam Houston Team have shown, by ^{133}Xenon studies, there is a significant adverse effect of steroids on small airway obstruction.

In spite of the permanent danger of infection of some injuries, the systematic use of antibiotics at the very first do not seem to us advisable, and, as B. Pruitt and other authors have shown, the use of prophylactic antibiotics may ultimately create more problems than benefits. In any case, their choice must be controlled, as long as it is possible, by protected bronchial samplings, carried out by fiberoscopy, trans-tracheal puncture being absolutely prohibited.

On the other hand, vaccines, useful in any gravely burned patient, seems here again strongly indicated. Although some authors advocate the use of bronchodilators, the concentration of circulating catecholamines or beta mimetics is sufficient to rule out their general use. Maybe they would be useful in an aerosol for inhalation?

We question its efficiency because of the large particle size, but they might be used to transport certain drugs the local effects of which would be beneficial in large bronchi.

Another category of drugs rarely mentioned includes neutralizing drugs or chelating agents of poisons formed in the blood. For example, hydroxocobalamine which is used when the cyanide level in the blood is above 60 micromol/liter and above all if there is an associated carbon monoxide poisoning.

Fibroscopy has a triple role. At the same time, it is a means of diagnosis, of observation and a means of therapy. On the therapeutics levels, it initially allows one to carry out bronchial washing (soot, caustic particles), alveolar washing — titration of the surfactant — and later on to remove an atelectasis; also to clear the bronchi and finally to follow the development of the injuries and the evolution of local flora.

At last, as far as respiratory kinesiotherapy is concerned, it need not be applied initially, it is useless for white lung, but its role must not be forgotten if the patient has an obstruction. The removal of an atelectasia may be carried out in many cases with kinesiotherapy alone, associated or not with fiberoptic endoscopy.

With a better understanding of the factors operative in primary respiratory lesions, and the development of improved methods to more adequately control respiratory damage, we can hope that pulmonary derangements associated with thermal injury will no longer be a major cause of mortality.